

RESPONSE TO SUPPLEMENTARY QUESTIONS
ARISING FROM IDSP REPORT NO.2 ON
OCCUPATIONAL EXPOSURE TO PCBs

Industrial Disease Standards Panel
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ARISING FROM IDSP REPORT NO. 2
OCCUPATIONAL EXPOSURE TO PCBs**

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January 10, 1990

Dr. Robert G. Elgie
Chairman
Workers' Compensation Board
2 Bloor St. E., 20th floor
Toronto, Ont.
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Dear Dr. Elgie:

Re: Occupational Exposure to PCB's
(IDSP Report No. 2)

By letter dated May 10, 1989, the Board posed certain questions arising from its consideration of the above IDSP Report. I am pleased to convey herewith the Panel's response, approved on January 10, 1990. The Panel requests that this document become a matter of public record at a time the Board deems appropriate, provided this is no later than the day on which the Board publishes its official response to IDSP Report No. 2.

Yours sincerely

J. Stefan Dupré
Chairman

JSD:dc



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January 10, 1990

MEMORANDUM TO: WORKERS' COMPENSATION BOARD

FROM: INDUSTRIAL DISEASE STANDARDS PANEL

RE: RESPONSE TO SUPPLEMENTARY QUESTIONS ON
OCCUPATIONAL PCB EXPOSURES

1.0 BACKGROUND AND SUPPLEMENTARY QUESTIONS

1.1 In December, 1987, the Panel issued its REPORT TO THE WORKERS' COMPENSATION BOARD ON OCCUPATIONAL EXPOSURE TO PCBs (IDSP Report No. 2). In that Report, the Panel found a probable connection between liver, biliary tract and gall bladder cancers and occupational exposure to PCBs (Finding 1). It also found no probable connection between any other site-specific cancers and occupational exposure to PCBs (Finding 2).

1.2 To assist the Board in its adjudication of liver cancer claims, the Panel provided two eligibility rules. The first rule recommended compensation when the claimant had at least 3 months of occupational PCB exposure, and at least 5 years of latency from first exposure to diagnosis (Eligibility Rule 1). A second rule was intended to address situations "of short, intense exposure to PCBs arising from sudden and unusual work circumstances such as PCB fires or explosions" (IDSP Report No. 2, p.7). The rule is quoted in its entirety below:

ELIGIBILITY RULE 2: WHERE DISPUTE ARISES IN THE APPLICATION OF ELIGIBILITY RULE 1 AND CIRCUMSTANCES PERMIT, CLAIMS ARISING FROM LIVER, BILIARY TRACT AND GALL BLADDER CANCERS AMONG WORKERS FOR WHOM THE BODY BURDEN OF PCBs AS MEASURED BY PCB BLOOD SERUM CONCENTRATIONS IS SIGNIFICANTLY HIGHER THAN THE NORMAL BACKGROUND DISTRIBUTION OF PCB BLOOD SERUM CONCENTRATIONS IN THE GENERAL POPULATION, BE COMPENSATED.

- 1.3 On May 10, 1989, the Board wrote the Panel requesting an elaboration of its Eligibility Rule 2. In particular, the Board requested answers to the following questions:

- Q1. What is the relevance of blood serum PCB levels to overall PCB body burden?
- Q2. What does the Panel consider to be a PCB level that is significantly higher than the normal background concentration found in the general population?
- Q3. What are the recommended procedures for sampling and analysis of PCB concentrations in blood serum?
- Q4. What is the clinical significance of "abnormal" PCB blood serum concentrations?
- Q5. What blood serum concentration level might be considered a potential health hazard for a worker to continue in his/her current job (i.e. PCB exposure)?

2.0 PANEL RESPONSE AND RECOMMENDATION

- 2.1 The Panel decided to respond to the first three questions only because the last two (on the clinical significance of "abnormal" PCB blood serum concentrations; and on worker removal criteria) were not material to the elaboration of Eligibility Rule 2. Accordingly, Panel staff prepared a report responding to these questions. The report is attached as Appendix A ('Serum PCBs in Normal and Exposed Populations', L.F. Smith, Nov. 6, 1989).
- 2.2 On the relevance of blood serum PCB levels to overall PCB body burden (the Board's Q1.), the report points out that PCBs are detectable in all organ systems of the body. However, the largest proportion are found in the adipose tissue compartment (the storage medium), and are also measurable in blood (the transport medium). The recent scientific literature shows that the overall PCB body burden can be determined from the serum PCB level when combined with measurements of serum lipids and total body fat estimates (Appendix A, p.4).
- 2.3 On the question of a PCB serum level that is 'significantly' higher than the normal background concentrations found in the general population (the Board's Q2.), the issue is complicated by the fact that observed PCB levels appear to drop quickly following removal from exposure. Given sufficient time (as little

as 4 to 6 weeks), PCB concentrations can decline to the point where they do not differ significantly from those for the general population unexposed beyond the food chain. For instance, one study showed that the levels of total PCBs in the adipose fat of previously exposed workers and unexposed workers were not significantly different. Moreover, the serum PCB levels for previously exposed workers were intermediate to those for currently exposed workers and workers unexposed beyond the food chain (Fait et al., 1989).

- 2.4 Even 'normal' levels of serum PCBs are subject to variability due to different stresses (e.g. major weight loss, pregnancy, etc.). Population levels for serum PCBs can range up to 30 ppb although observed means are usually below 10 ppb. The corresponding adipose fat PCB concentrations are about 100 to 150 times the values for blood, or about 1500 ppb (Appendix A, p. 4-7).
- 2.5 The question of higher than normal PCB serum levels is complicated further by the vagaries in measurements (the Board's Q3.). Serum levels below 20 ppb can be considered as "possibly confounded by other organochlorine compounds". Levels above 20 ppb must be confirmed by an appropriate method (viz. gas chromatography coupled with mass spectrometry); as well as by the use of a single laboratory to ensure consistency of measurements and standardization of procedures (Appendix A, p. 7-10).
- 2.6 For all of the above reasons, the Panel has decided to move away from blood serum assays when considering instances of intense PCB exposure arising from sudden and unusual work circumstances such as fires, explosions or accidents. Instead, the Panel recommends the Board's adoption of the following revised Eligibility Rule 2:

ELIGIBILITY RULE 2: NOTWITHSTANDING THE MINIMUM EXPOSURE DURATION PERIOD IN ELIGIBILITY RULE 1, CLAIMS ARISING FROM LIVER, BILIARY TRACT AND GALL BLADDER CANCERS AMONG WORKERS SHALL BE COMPENSATED WHERE THERE IS EVIDENCE OF INTENSE EXPOSURE TO PCBs SUCH AS THAT OCCASIONED BY FIRE, EXPLOSION OR ACCIDENT.

- 2.7 This Memorandum has been approved by all Panel Members present at IDSP Meeting No. 34 on January 10, 1990 (Panel Members Buck, Chong, Dupre, Hess, Miller and Stevens).

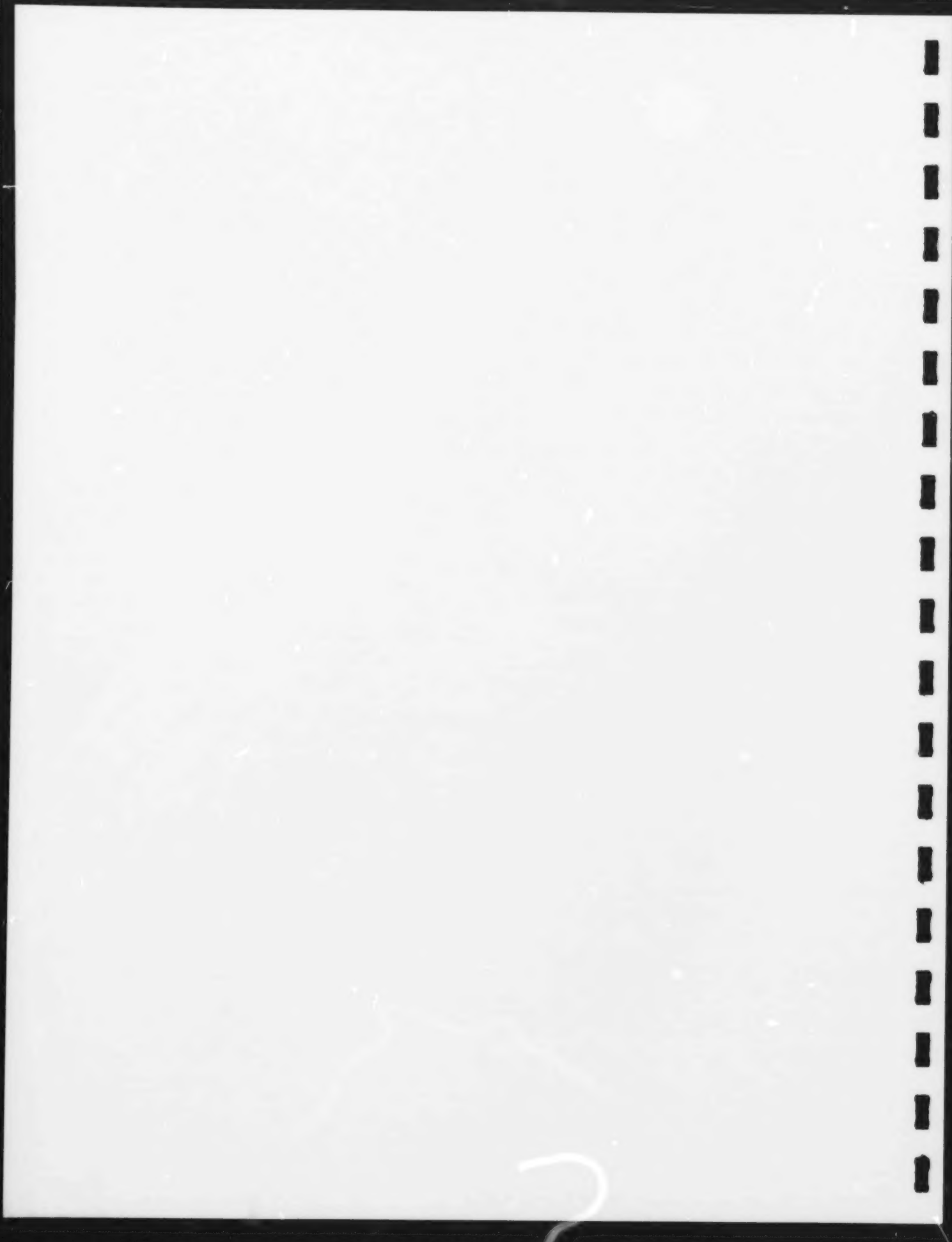


APPENDIX A

SERUM PCBs IN NORMAL AND EXPOSED POPULATIONS

L.P. Smith

Industrial Disease Standards Panel
Nov. 6, 1989



SERUM PCBs IN NORMAL AND EXPOSED POPULATIONS

L.F. Smith

Industrial Disease Standards Panel
November 6, 1989**Introduction:**

The Industrial Disease Standards Panel published its Report to the Workers' Compensation Board on Occupational PCB Exposure in December, 1987 (IDSP Report No. 2). In this report, the Panel recommended two eligibility rules for the compensation of claims among workers with cancers of the liver, biliary tract or gall bladder and with opportunity for exposure to PCBs in the workplace. The first eligibility rule required at least three months of employment in a workplace with PCB exposure and at least a five year latency from the time of first exposure.

The Panel recognized that brief, intense exposures (e.g. in fires or explosions) may occur and that the worker in such a circumstance might not meet the minimum exposure duration (three months) and or the latency requirement (five years) required under Eligibility Rule 1. Thus, the Panel recommended that where a dispute arises in the application of Eligibility Rule 1 and circumstances permit, such claims be compensated if it can be demonstrated in the worker that **'... the body burden of PCBs as measured by PCB blood serum concentration is significantly higher than the normal background distribution of PCB blood serum concentrations in the general population.'**

In response to these Panel recommendations, the Workers' Compensation Board has asked the Panel to answer the following questions (Letter from R.G. Elgie dated May 10, 1989):

1. What is the relevance of blood serum PCB levels to overall PCB body burden?
2. What does the Panel consider to be a PCB level that is significantly higher than the normal background concentration found in the general population?
3. What are the recommended procedures for sampling and analysis of PCB concentrations in blood serum?
4. What is the clinical significance of "abnormal" PCB blood serum concentrations?

5. What blood serum concentration level might be considered a potential health hazard for a worker to continue in his/her current job (i.e. PCB exposure)?

The present review addresses the first three questions posed to the Panel by the Board.

A dispute could arise in the application of the eligibility rules where a worker had 'opportunity for exposure' (e.g. from a fire, explosion, spill, transient excursions of the threshold limit value, etc.) but where no monitoring of serum PCB level was available at or immediately following the time of the exposure. Such a worker might be subjected to a risk due to the added burden of PCBs in the body caused by a high, but transient exposure which would not meet the three month requirement of Rule 1.

CONSIDERATION OF THE BOARD'S QUESTIONS:

1. What is the relevance of blood serum PCB levels to overall PCB body burden?

This question raises two issues:

- . How does blood PCB concentration relate to body burden?
- . How does one measure the PCB body burden?

PCBs comprise a group of compounds containing a biphenyl nucleus with different numbers of chlorine atoms attached. Biphenyl molecules with different numbers of chlorine atoms attached are known as congeners of polychlorinated biphenyls. There are 209 such compounds. Two PCB molecules containing the same number of chlorine atoms in different locations of the biphenyl molecular ring may differ considerably in their behaviour. Such (nearly identical) molecules are referred to as isomers. PCB isomers are distinguished by the location of the atoms of chlorine (structural isomers) or by the spatial relationship of the chlorine atoms within the molecule (stereochemical isomers).

Some level of PCBs can be detected in all organs, the largest proportion being found in the adipose tissue compartment (fat). PCB concentration can be measured in blood (the transport medium) and in fat (the storage medium). However, blood is readily accessible for measurement while fat can only be obtained through an invasive procedure such as suction of subcutaneous fat or excision of adipose tissue during surgery

for another reason.

PCBs in the blood are in dynamic equilibrium with stored (adipose tissue fat) PCBs. The 209 congeners of PCB have different half-lives in the body. Bertazzi reported an approximate half-life for PCB blood concentrations of Aroclor 1254 of about ten years (Bertazzi, P.A. in IDSP Report no. 2, 1987). One infers thereby that the PCB body burden has on average also fallen by 50% in ten years. From studies in mice, it is known that some congeners are cleared relatively quickly while others persist. Elimination studies of working populations usually consider men only. The half-life in women may vary significantly from men. Yakushiji et al. reported a 9.2% decrease per year in Japanese women with occupational exposure (Yakushiji et al., 1984). This rate was similar to the elimination rate in their offspring (10% per year) after adjustment for growth. The elimination of PCBs in females is aided by pregnancy and lactation which provide added excretion pathways through the milk and placenta. Females at all ages tend to have lower serum PCB levels than males (Kreiss and Roberts, 1982).

The relationship between the PCBs in adipose tissue fat, blood, and vital organs may vary considerably (Matthews, 1984). This relationship is influenced by exposure level, age, sex, size of adipose tissue fat compartment, length of exposure, and time of exposure (current vs. past) (Kimbrough, 1985). This relationship also varies with the particular PCB congener (i.e. degree of chlorination) (Fait et al., 1989). Variations of blood PCB level occur depending on physiological state: fasting, weight loss or weight gain, pregnancy, breast feeding, etc. Blood PCB level is associated with age, serum lipids, and (contaminated) fish consumption among many other variables (Kreiss, 1985; Schwartz et al., 1983).

Brown and Lawton (1984) examined 173 capacitor workers exposed to Aroclor 1254, 1242 and 1016. Wolff et al. reported the fat-plasma distribution coefficients of 35 PCB isomers in 26 of these 173 capacitor workers (Wolff et al., 1982). Wolff found that the ratio between adipose fat and blood concentrations varies from under 100:1 to over 300:1 (average 190:1).

Neutral lipids (i.e. neutral fats) or glycerides are a major component of storage fat (in adipose tissue fat cells). Triglycerides are one portion of the neutral fat content. Cholesterol is a non-glyceride component of fat found in serum either "free" or attached to glycerides. Brown and Lawton (1984) empirically found that PCBs partition between serum and adipose tissue fat in direct proportion to their content of neutral lipids, and not to their content of cholesterol.

In particular, Brown and Lawton examined a capacitor worker with hyperlipemia (high serum lipid concentrations) from the same group of workers reported above. Three congeners of PCBs were measured before and after a weight-reduction diet resulting in 4.5 kg weight loss. The serum PCB levels changed as follows: Aroclor 1244 - 1195 to 774 ppb; Aroclor 1254 - 57 to 33 ppb; Aroclor 1260 - 30 to 19 ppb. Serum triglycerides changed from 675 to 218 mg%; cholesterol from 236 to 240 mg%. These results demonstrate the dependence of serum PCB level upon the serum lipid concentration (i.e. neutral fat concentration).

These authors demonstrated the following model of PCB distribution applicable to humans. In the equilibrium state, the ratio of serum lipid PCBs to that of the total serum neutral lipids or glycerides is equal to the ratio of adipose fat PCB level to the neutral fat content of the tissues. This equilibrium relationship can be applied to the individual. If an estimate of total body fat is available, the total body burden of PCBs can be estimated roughly from measurement of serum PCBs and serum lipid levels alone. That is, the total body burden of PCBs can be estimated by multiplying the ratio of serum PCB concentration to serum lipid concentration by the total body fat content.

In summary, to answer question 1, the best estimate of PCB body burden is obtained by determining the concentration of serum lipid PCBs and the estimate of total body fat. Serum PCB level is the nearest indirect measure of body burden one can obtain when combined with serum lipids and total body fat.

2. What does the Panel consider to be a PCB level that is significantly higher than the normal background concentration found in the general population?

Fait et al. (1989) reported the levels of total PCBs (and PCB congeners) in the serum and adipose fat of past and present transformer workers and an unexposed comparison group. The results of some of their findings are presented in Tables 1, 2, and 3. They noted that the total PCB levels in adipose fat of previously exposed workers and unexposed workers were not significantly different. They concluded that there had been significant elimination over time. Therefore, the level of PCB in fat might not be discriminating enough to distinguish a previously exposed worker from 'background' if sufficient time has elapsed since the exposure of interest.

Given the number of variables which affect the level of PCBs in the blood of an individual and the vagaries of measurement (discussed separately below), normal values are probably best interpreted from the analysis of blood from large samples of

the general population not exposed beyond the food chain. Also given that adipose tissue levels may not be available for the purpose of interpretation of a blood PCB level in an individual, then blood (preferably serum) PCB is the best estimate of that individual's body burden provided there are no extraordinary stresses such as major weight loss, pregnancy, etc., or current extraordinary exposure. One must consider the time when 'extra' exposure to PCBs occurred in interpreting whether an individual falls outside the range of 'normal'. For example, immediately after a single, acute exposure (such as from a spill or transformer fire), blood PCB rises only to fall to pre-exposure level in about four to six weeks (Luotamo et al., 1985; Elo et al, 1985; Wolff, 1985).

As the body eliminates significant amounts of PCBs over time, a level of PCB in fat (whether calculated by formula from serum lipids, serum PCBs and total body fat) or measured directly in fat (obtained by biopsy) only reflects current body burden which may not differ from 'background' and may not reflect the body burden at the time that the exposure of interest occurred. A demonstrated rise in serum PCB level with a demonstrated fall to pre-exposure level within six weeks of exposure represents the best evidence of acute exposure.

PCBs are a mixture of compounds with various degrees of chlorination, toxicity and retention in the body. The general population is exposed to PCBs primarily through the food chain. The earliest exposure occurs through mother's milk. Continued exposure is assured through milk, eggs, fish and meat. Additional exposure can occur through the consumption of marine mammals, a custom prevalent in certain population groups such as the Inuit. An individual exposed through an occupation may have an added body burden of PCBs above a background. If the background level can be estimated, then the difference between that and the measured burden in the individual would reflect the occupational component.

What test for an individual worker, therefore, could discriminate between a previous significant exposure and no exposure beyond the food chain?

The level of PCB as measured in blood or fat, and the corresponding total body burden in the general population reflect the background for those not exposed beyond the food chain. The literature was reviewed to establish the state of knowledge on normal background levels for blood and fat PCBs. Since PCBs are stored in fat, all variables being equal, total body burden would be reflected in their concentration in that tissue.

Blood PCBs

Several environmental accidents have provided the opportunity to study the level of PCBs in the blood of a large number of individuals. In 1973, the fire retardant Flamemaster (composed of PBBs or polybrominated biphenyls), made by the same company as Nutrimaster, a cattle feed additive (magnesium oxide), was accidentally mixed into cattle feed. This mixup resulted in the contamination of a large number of cattle with PBBs and consequently, of thousands of people in the state of Michigan. The followup of this large cohort exposed to PBBs has also included measurements for PCBs (Kreiss et al., 1985). The measurements for PCBs reflect background values. Control values were sought when acute exposures occurred (fires, explosions) and these values are also presented.

Table 1 lists all the reviewed studies by author, year, number of subjects, medium analyzed, and levels found. Table 2 contains data from the same studies on blood (Table 2A) and fat (Table 2B) PCB levels for control groups unexposed beyond the food chain.

Kimbrough (1985) reports that data from the Centers for Disease Control in the United States show that the mean PCB concentration in the serum of individuals not exposed beyond the food chain is between 5 to 7 pbb, and that some individuals may have higher levels without any evidence of extraordinary exposure. Safe (1987) refers to levels from <10 to 15 pbb in the general population unexposed beyond the food chain. Kreiss et al. (1982) have reported the largest population of individuals unexposed beyond the food chain. The highest level found was 57 ppb, the arithmetic mean 7.7 ppb and the geometric mean 6.4 ppb (males: 7.1 ppb; females: 5.7 ppb). Ninety-five percent of this population unexposed beyond the food chain had a serum PCB level of less than 20 ppb (Kreiss, 1985). This large sample of North Americans unexposed beyond the food chain probably reflects the best estimate of "background". The average blood PCB level for this group unstandardized for age and sex is 10 ppb (Kreiss, 1985). This is supported by a more recent survey of a population applying for work tested as part of the pre-employment assesement. This group, the second largest U.S. sample, showed a mean blood PCB level of 5 ppb [$\pm$ std. dev'n. of 4.25 ppb] with a range from 1 to 37 ppb (Sahl et al., 1987).

In a smaller sample of 58 persons, Fait et al. found that 75% had a serum PCB level below 25 ppb. The several reports of residents living around an area with PCBs in soil have generally not demonstrated a range of values different from those reported by Kreiss et al. or others. Average levels in

such populations with opportunity for exposure have been close to or lower than those unexposed beyond the food chain (Jan and Tratnik, 1988; Smith, 1986). Non-randomly selected population samples have been tested for PCB concentration in the blood. At least a dozen such groups unexposed beyond the food chain report a range of PCB in blood of 0 to 60 ppb, a distribution of values similar to those of the largest sample (Kreiss and Roberts, 1982; Kreiss, 1985).

US CDC data show that the adipose tissue concentration of PCBs is about 100 to 200 times as high as the serum level (500 to 1400 ppb) (Kimbrough, 1985).

Several transformer accidents have afforded opportunities to study PCBs in the blood of acutely exposed groups. The values reported in Table 3 are among workers acutely exposed during transformer spills or fires. Some acute exposures were superimposed on chronic exposure.

Emmett studied 55 current and past PCB-exposed transformer workers. Serum PCB and adipose fat PCB levels were determined. Adipose fat and serum PCB were higher in currently exposed workers (2100 ppb mean in adipose fat; 12.2 ppb in serum) than in past exposed (830 ppb mean in adipose fat ; 5.9 ppb in serum) or never exposed (600 ppb mean in adipose fat; 4.6 ppb in serum) (Emmett, 1985). However, there was no difference between the past exposed group and the unexposed group for either serum or fat.

Fait et al. studied a group of past and present transformer repair workers and a comparison group. A pattern similar to Emmett's was found. Serum PCBs were highest in the currently exposed group and lowest in the never exposed while the past exposed was intermediate. Adipose fat PCBs in the currently exposed was much higher than in the other two groups but the distribution of values in the past exposed and unexposed was similar (Fait et al., 1989). Both of these well-conducted studies suggest that serum PCB alone, or adipose fat PCB alone will not distinguish the past exposed from the never exposed. These values may be applied to population or group averages (means) rather than to individuals.

Even though the equilibrium model proposed by Brown and Lawton can help in calculating body burden with certain parameters in hand for an individual (total body fat, serum lipids and serum PCB), the result may not reflect an elevated body burden if the person has not had exposure to PCBs for a number of years. Table 3 shows values of PCBs in the blood of acutely and chronically-exposed groups which illustrate the points made by Emmett and by Fait et al.

In summary, the literature points to a population range of

PCBs in blood of less than 30 ppb with occasional individuals exceeding this level. The unstandardized population mean is below 10 ppb. The corresponding values for adipose fat PCB level is in the range of 100 to 150 times that of the blood, or about 1500 ppb.

3. What are the recommended procedures for sampling and analysis of PCB concentrations in blood serum?

Laboratory methods:

In order to ensure reliable measurements, the sampling protocol for PCBs, as for many other materials, must be rigorous from the taking of the blood through all of the analytic steps performed. Whole blood is collected from the donor, allowed to clot, and the resulting serum (whole blood minus cellular components and labile clotting factors) is analyzed for PCBs. Plasma (whole blood free of cellular elements only) is sometimes used for analysis; fat is rarely available. Whole blood (with all its cellular elements) is used by some laboratories. Analysis of whole blood requires that the sample be homogenized before it is subjected to analysis. The tube in which blood is collected must be clean and free of the contaminant to be tested. Blood for PCB should be drawn by glass syringe and placed into a glass tube with no anticoagulant. This allows the blood to clot and clot retraction to take place, leaving clean serum behind. Some laboratories may still use whole blood or plasma as the medium for analysis but serum is generally preferred. The Ontario Ministry of Labour Occupational Health Laboratory will accept vacuum collecting tubes (Vacutainer[®]) without anticoagulant for analysis. They prefer that the sample be transferred to a teflon cap test tube. The use of plasma presupposes the use of an anticoagulant in the collection of the blood specimen and the possible need for additional clean-up during the extraction procedure discussed below.

Analytic methods now available generally use a four step procedure:

1. extraction of the PCBs from the medium (serum, plasma, fat, etc.);
2. cleanup of the sample;
3. separation of the PCBs from the extract; and
4. quantification with or without confirmation.

Problems which can arise in the handling of samples can be grouped into contamination, loss, desiccation and alteration

(Albro, 1979). Errors can occur in the extraction process in that recovery from the sample may vary. Each laboratory must carry out its own quality control studies to ensure that the extraction process produces consistent recovery throughout all of the relevant concentrations for PCBs in the different media. These results should be reproducible. Similarly, the cleanup procedure must demonstrate consistently reliable results.

Extraction of PCBs:

PCBs are extracted from the fat in the sample by homogenizing the sample (in the case of tissue, fat or whole blood) and mixing with a solvent such as hexane, alcohol, chloroform, acetone.

McKinney et al. reported the most extensive study of laboratory methods using milk, serum and fat. They reported a recovery of 90 to 100% of radioactively-labelled Aroclor 1254 from serum (McKinney et al., 1984). Other reliable laboratories carrying out similar quality control procedures report a recovery of $84.6 \pm 11.4\%$ (Luotamo et al., 1985). The Ontario Ministry of Labour Occupational Health Laboratory uses analytic methods similar to those reported throughout the literature (Dr. Mark Nazar, personal communication, June 29, 1987). Their PCB recovery from serum is about 90% (Dr. Virindar S. Gaiind, personal communication, October 11, 1989).

Clean-up:

This process involves the removal of fat and the separation of PCBs from other organochlorine compounds. The literature reports various ways of cleaning up the sample by the use of acetonitrile or dimethylformamide, or treatment with strong sulfuric acid or ethanolic potassium hydroxide. PCBs can be separated from organochlorine pesticides by column chromatography using Florisil, silica gel, or charcoal columns. Different laboratories tend to use different types of columns and materials for this step.

Chromatographic separation of PCBs:

The extract produced after cleanup is then presumed to contain the PCBs. This extract is submitted to gas-liquid chromatography and electron capture detection. Peaks (on graph paper) of different PCBs are obtained. Enhanced visual separation of these peaks occurs with by prior fractionation of the extract through charcoal or glass capillary columns.

Quantification of PCB content:

The response of the electron capture detector is affected by the degree of chlorination in the PCB sample. Degree of chlorination may vary with degradation (or metabolism) of the product. It is difficult then to match the obtained peaks of PCB metabolites with the peaks created by standard preparations of PCBs. Discrepancies in the reading of these peaks may occur.

Confirmation of identity of the PCBs:

Samples may then be subjected to analysis with mass spectroscopic methods which can determine very low concentrations of the PCBs.

Reference standards for all 209 PCB congeners are available now. This makes it easier to identify more accurately a variety of PCBs making the estimate of total PCBs also more accurate. However, most studies reported in the literature did not have the benefit of the now available standards so that comparison of some older reported values with current more accurate ones for this as well as other reasons is not advisable.

The Occupational Health Laboratory of the Ontario Ministry of Labour will confirm results of gas-liquid chromatography with gas chromatography-mass spectroscopy (GC-MS) if the quantification procedure indicates levels in blood above 20 ppb. The detection limit of the GC-MS is 20 ppb. Values lower than 20 ppb are considered "unconfirmed, possibly PCBs".

Laboratory comparisons:

Several investigators have pointed out that current methods used to measure PCBs may be relatively imprecise (Albro, 1979; Lawton and Ross, 1986; Burse et al., 1983a, 1983b). Lawton and Ross found considerable imprecision which they ascribed to random errors, interlaboratory variations in the procedure, and methods of data reporting (Lawton and Ross, 1986). Burse et al. (1983a) reported considerable variability in a comparison among twenty-five laboratories. A subsequent study of 44 laboratories demonstrated that interlaboratory variability of serum PCB measurements still remained but could be reduced by standardizing the methods (Burse et al. 1983b).

In conclusion, the literature is generally critical of the accuracy of the analytic methodology for PCBs. Levels below 20 ppb can be considered as "possibly confounded by other organochlorine compounds". Levels above 20 ppb must be

confirmed as due to PCBs by an appropriate method such as gas chromatography coupled with mass spectroscopy. Comparability of results is aided by the use of a single laboratory with consistent results using the same medium (i.e. serum), and standard methods with high level of recovery and reliability. These limits must be considered in the interpretation of blood serum PCB levels especially in the lower concentration range (below 20 ppb). Some authors recommend the use of a single laboratory with an acceptable but stable extraction level even if the extraction is not very high rather than a high but inconsistent extraction. The literature warns that the differences found among laboratories merit attention when results of different studies are compared with each other and "... when considering the use of a specific serum PCB tolerance limit as a basis for administrative action" (Lawton and Ross, 1986).

TABLE 1

STUDIES REPORTING PCB LEVELS
IN WHOLE BLOOD, SERUM, PLASMA AND FAT

AUTHOR	YEAR	STUDY GROUP	CONCENTRATION IN PPB [+/ -SD] [RANGE]	CONTROLS	CONCENTRATION IN PPB [+/ -SD]	MEDIUM
Bush	1984	101 women post-birth	3.5 [+/ -1.1]			whole blood
Baker	1980	89 sewage sludge users	17.4	18 capacitor workers 19 fam. mem. of workers 22 commun residents	75.1 33.6 24.4	serum
Steinberg	1986	100 residing near electrical equipment plant	[4-75]	none	--	serum
Sahl	1987	738 pre- employment		5 [+/ -4.25] [1-37]		serum
Stark	1986	transformer explosion 12 Utility Co. 16 firemen 14 policemen 10 bldg tents.	9.7 [+/ -7.4] 6.8 [+/ -2.8] 6.6 [+/ -2.3] 4.8 [+/ -1.8]	11 utility co. 22 firemen 22 policemen 6 tenants	7.1 [+/ -2.7] 7.4 [+/ -3.1] 6.4 [+/ -2.2] 5.9 [+/ -1.9]	blood

TABLE 1 (CONT'D.)
STUDIES REPORTING PCB LEVELS
IN WHOLE BLOOD, SERUM, PLASMA AND FAT

AUTHOR	YEAR	STUDY GROUP	CONCENTRATION IN PPB [$\pm$ -SD] [RANGE]	CONTROLS	CONCENTRATION IN PPB [$\pm$ -SD]	MEDIUM
Chase	1982	86 men with direct contact with transformer fluids	33.4 5600	15 same co. no contact	14.2 1400	plasma fat
				19 other co.	12 1300	plasma fat
Fischbein	1979	capacitor plant	low homologues	high homologues		plasma
		43 high exposure	266 [$\pm$ -328]	82 [$\pm$ -128]		
		62 medium "	171 [$\pm$ -361]	25 [$\pm$ -25]		
		158 low "	73 [$\pm$ -81]	41 [$\pm$ -75]		
		26 retired	80 [$\pm$ -80]	84 [$\pm$ -120]		
Maroni	1981	Plant A	Pyralene3010	Plant B	Apirolino	blood
		48 current exp.	377 [$\pm$ -258]	12 current exp.	200 [$\pm$ -146]	
		16 past exp.	292 [$\pm$ -161]	1 past exp.	104	
		3 no exposure	110 [$\pm$ -31]			
Fait	1989	35 transformer repair workers	43.7 3180	56	16.1 821	serum fat
		17 former repair workers	30 888			
Acquavella	1989	205 capacitor plant workers	18.2 [$\pm$ -2.88]	none	--	serum

TABLE 1 (CONT'D.)

STUDIES REPORTING PCB LEVELS
IN WHOLE BLOOD, SERUM, PLASMA AND FAT

AUTHOR	YEAR	STUDY GROUP	CONCENTRATION IN PPB [±SD] [RANGE]	CONTROLS	CONCENTRATION IN PPB [±SD]	MEDIUM
Fitzgerald	1986	482 exposed in transformer fire	6.9 [±3.52]	none	--	serum
		90 low exposure	6.46			
		76 medium "	6.59			
		92 high "	7.84			
Skaare	1988	43 women post-birth	10 [±4]			serum
Stehr-Green	1988	exposure to 12 waste sites	10.9 [3-75]	8	5.9 [4-13]	serum
Kreiss	1982	PPB exposed to PBBs, not PCBs		1631	6.4	serum
Rogan	1986	880 women post-birth	9.06			serum
Jan	1988	10 residents along Krupa river (waste site)	155	19 1-3km away from river 4 >20km away from river	11 5	blood

TABLE 2A

BLOOD PCB IN 'NORMAL POPULATIONS'
(UNEXPOSED TO PCBs BEYOND THE FOOD CHAIN)

AUTHOR	N	CONCENTRATION IN PPB: [RANGE], [+/- SD] OR DISTRIBUTION	DESCRIPTION OF STUDY GROUP
Kreiss 1982 ¹	1631	6.4 95% <20 [1-57]	all ages, USA population
Chase 1982 ²	19	12 [10-27]	worker controls
Stark 1983 ³	11	7.1 [+/-2.7]	utility company controls
	22	7.4 [+/-3.1]	firemen controls
	22	6.4 [+/-2.2]	policemen controls
	6	5.9 [+/-1.9]	tenant controls
Steinberg 1986 ⁴	100	[4-75]	resident controls
Jan and Tratnikk 1988 ⁵	4	5	resident controls
Skaare et al. 1988	14	10 [+/-4]	Swedish women (c-sections)
	20	10 [+/-7]	Swedish women (vaginal delivery)
Fait 1989 ⁶	56	50% <16.1 25% < 8.2 75% <24.4	controls
Takenaka et al. 1988 ⁷	32	3.34 [+/-0.41]	'normal subjects' Japanese controls

1. Michigan PBB cohort also examined for PCBs. Arithmetic mean 7.7 ppb. The results presented include all ages.
2. Transformer explosion which exposed utility company workers, firemen, policemen and building tenants. Each group was tested; corresponding values for the controls shown here.
3. Transformer fire
4. Controls for residents living near a transformer plant.
5. Controls living 20 km away from a community in proximity to a contaminated sediments in the Krupa River (Yugoslavia).
6. Male workers in a transformer repair facility never exposed to PCBs.
7. Takenaka et al. used whole blood for analysis.

TABLE 2B

PCB CONCENTRATION IN ADIPOSE FAT OF THE GENERAL POPULATION

AUTHOR	N	CONCENTRATION IN PPB		DESCRIPTION OF STUDY GROUP
		[+/- SD] OR DISTRIBUTION	[RANGE]	
Fait 1989	56	50% < 821 25% < 708 75% < 1970	[<708-1970]	controls
Chase 1982	19	1300	[1000-2000]	controls for trans- former workers with exposure
Takenaka et al., 1988	30	837 [+/-74-]		'normal Japanese'
Skaare et al. 1988	16	662 [+/-401]		pregnant Swedish women

TABLE 3

PCBS IN BLOOD OF ACUTELY AND CHRONICALLY EXPOSED GROUPS

AUTHOR	N	CONCENTRATION IN PPB: [RANGE], [+/- SD] OR DISTRIBUTION	DESCRIPTION OF STUDY GROUP
Baker 1980 ¹	89	17.4	sewage sludge workers
Brown and Lawton, 1984	173	[12 - 392]	current capacitor workers
	173	[4 - 103]	Aroclor 1242;
	144	[4 - 29]	Aroclor 1254; Aroclor 1260.
Stark 1986 ²	12	9.7 [+/-7.4]	utility company workers
	16	6.8 [+/-2.8]	firemen exposed during a fire
	14	6.6 [+/-2.3]	policemen attending a PCB fire
	10	4.8 [+/-1.8]	residents of the building with a transformer fire
Fitzgerald 1986 ³	482	6.9 [+/-3.5]	exposed in a transformer fire
Takenaka et al. 1988	27	7.28 [+/-0.82]	Type A Yusho patients
Fait 1989 ⁴	35	50% <43.7	current transformer workers
		25% <20.1	
		75% <78.9	
	17	50% <30	past transformer workers
		25% <5.8	
		75% <60.7	
Acquavella 1989 ⁵	205	18.2 [+/-2.8]	capacitor plant workers

NOTES:

1. Exposure is low-level and longstanding.
- 2,3. Exposure is acute.
4. The groups studied include 35 transformer repair workers currently exposed to PCBs, 17 previously exposed transformer repair workers and 56 comparison never-exposed workers.
5. Capacitor plant workers only.

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